

The NCVDLs REPORT



Veterinary News and Information From North Carolina's Diagnostic Laboratories



North Carolina Department of
Agriculture and Consumer Services
Steve Troxler, Commissioner

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Holiday Closings...
September 3, 2012
November 12, 22 and 23, 2012
December 24, 25 & 26, 2012
January 1, 2013
Our laboratories will be closed on
the above listed days.

Please e-mail
NCVDL@ncagr.gov with any
comments and/or suggestions
concerning The NCVDLs
Report
Editor - Dr. David Drum

Message from the Director

The good news and the bad news. The NC General Assembly has just ended its legislative session and was successful in overriding Gov. Beverly Perdue's veto of the state budget. This paved the way for the first pay raise for state employees and retirees since 2008. State employees will also receive five extra days of annual leave. The bad news is that state agencies have been asked to reduce their operating budgets by 2%. Enough said.....We will continue to tighten our belts and assure that client services will not suffer the consequences.

A renewal inspection of the Rollins Biosecurity Level 3 (BSL3) Laboratory was conducted by auditors from the Centers for Disease Control (CDC) in January, 2012. The Rollins BSL3 facility is a registered entity with clearance to possess select agents which are biological agents and toxins that could pose a severe threat to public and animal health, or to plant health and plant products. Nationwide there are over 300 entities that are registered with and inspected by the CDC Select Agent Program. All aspects of the registration are managed by the program, including amendments to the registration; approval of transfers of select agents; and investigation of reports of theft, loss, or release of select agents. Entities are inspected every three years to ensure compliance with Select Agent Regulations. These assessments allow inspectors to confirm that the appropriate safety and security measures are in place, as well as, ensure that laboratorians are adequately trained. After the rigorous multi-day audit, I am pleased to announce that our registration was renewed for another 3 years.

As always, it is a pleasure to serve our stakeholders and we welcome your feedback.

Sincerely,

Karen W. Post DVM, MS



Feature Article

By Dr. Jennifer Haugland

Lead Toxicosis in a group of Beef Cattle

Five mixed breed beef calves 4-5 months of age out of 18 calves died over a 5-6 week period. The second dead calf was seen circling prior to drowning in the pond but the remaining calves were found dead with no prior clinical signs. The owner stated there was nothing in pasture that could be toxic (i.e. no equipment or batteries). The second and fifth dead calves were necropsied at the Rollins Laboratory 4 weeks apart. The only findings from the first examined calf were mild hepatitis and coccidiosis. Aerobic, salmonella, and Listeria cultures did not yield significant results. A moderate amount of Eimeria oocysts were found in feces. Brain was negative for rabies and there was no evidence of polioencephalomalacia or other neurological disease. Nervous coccidiosis was considered a differential although there was not a large amount of coccidia oocysts in the feces. Testing of tissues for heavy metal levels was recommended since lead toxicity was possible. A fifth calf died 4 weeks later with no prior clinical signs and was necropsied. Additional history obtained was that the herd of cows and calves, 2 donkeys and a horse were moved to the current pasture a few days before the first calf died and had not been moved since. No significant gross lesions were found in the fifth calf. Since toxicity was likely, the owner was strongly encouraged to ask a veterinarian to examine the herd and to walk around the pasture looking for potential toxins (metal, plant, pesticide, etc), which would allow us to focus on which type of toxin testing to request. A local veterinarian did walk the pasture the same day and reported that in the pasture there were several old tenant houses, an old hog barn, an old hog lagoon, and old batteries were found by the hog barn. It appeared that cattle had been around the batteries and had been in one of the houses. Liver and kidney from the 2 submitted calves were submitted to the Pennsylvania Animal Diagnostic Laboratory for heavy metal testing.

Heavy Metal Screen * results reported on a wet weight basis	1 st calf Liver	2 nd calf Liver	Normal reference range-liver	1 st calf Kidney	2 nd calf Kidney	Normal reference range – kidney
Arsenic ppm	<0.05	<0.05	0.02-0.13	<0.05	<0.05	0.02-0.20
Cadmium ppm	<0.02	<0.02	0.01-0.50	<0.02	<0.02	0.01-0.50
Lead ppm	6.21	11.5	0.04-0.50	264	6.35	0.04-1.00
Selenium ppm	<0.150	0.179	0.25-0.50	0.186	0.273	1.00-2.50
Thallium ppm	<0.05	<0.05	<0.05	<0.05	<0.05	0.05-0.06

Diagnosis was lead toxicity. Lead tissue levels greater than 10 ppm are considered diagnostic for lead toxicity. Lead is the most common cause of toxicity in cattle. Clinical signs of acute lead toxicity in cattle involve central nervous derangement and may include depression, hyperesthesia, muscle fascicula-

Feature Article continued

tions, ataxia, vocalizing in an altered voice, blindness, head pressing, charging, circling, recumbency, convulsions and death within 12-24 hrs. Death without clinical signs, like in this case, is also very common. Necropsy examination may reveal reddened or ulcerated gastrointestinal mucosa or constipation, but usually no gross lesions are present. Textbooks report that microscopic lesions may be present but they are very rare in the cases seen at NCVDLs. Diagnosis is made by blood or tissue analysis. Common sources of lead are car batteries, paint, oil, lubricants, machinery grease, trailer batteries, linoleum, roofing felt, caulking compounds, putty, roofing tiles, and lead arsenate defoliants (Gypsine and Soprabel). Grazing cattle on pastures that contain old buildings, piles of trash or any equipment is a practice that is highly associated with lead toxicity. The younger cattle are often the only ones affected because they absorb more lead at rates of 2-10% more than adults and they seem to be more likely to play with and consume lead contaminated substances. Also lead absorption is enhanced by a milk diet.

Lead ingestion also results in aberrations of other minerals, for example, long term exposure diminishes the absorption of selenium by as much as 26%. Lead toxicity in this case may have contributed to the deficient levels of selenium in both calves.

Lead toxicity should be highly suspected when predominantly young cattle die suddenly with neurological or no clinical signs after recent introduction to a new pasture that contains any amount of equipment, trash, or old buildings.

References:

Puls, R: *Mineral levels in Animal Health*, ed 2, British Columbia, 1994, Sherpa International

Short Cuts

COMPANION ANIMAL

Canine

Acute respiratory distress syndrome (ARDS) in a Mexican Hair-less Dog

According to the history provided, a 7 year-old, Mexican- Hairless dog began an acute onset of harsh coughing on a Friday afternoon. The dog was treated with Lasix, antibiotics, steroids, and oxygen therapy with little improvement at a local emergency clinic. On Sunday, the dog was transferred to an internal medicine specialists hospital for evaluation. On physical examination, the dog was BAR, had severe crackles throughout all lung fields, making heart auscultation difficult. The submandibular lymph nodes were mildly enlarged. The respiratory effort and rate was markedly increased. Thoracic radiographs showed severe diffuse unstructured interstitial lung pattern present on right lateral view. The cardiac silhouette appeared to be within normal parameters, but pulmonary vessels cannot be critically assessed

COMPANION ANIMAL, CONTINUED

due to overlying pulmonary disease. Mild aerophagia was noted. Differentials for the lung patterns included hemorrhage, DIC, infection (viral- distemper, influenza, bacterial- hematogenous, sepsis), ARDS, and inhalent irritants. A blood chemistry panel was unremarkable. The CBC showed elevated WBCs (41,000 [5000-16,000 reference range]), with neutrophils and monocytes significantly elevated. A brief bronchoscopy was performed, no foreign bodies or tumors were observed in the airways. A mild amount of exudate was noted, and the larynx was normal in appearance and movement. A sterile endotracheal lavage was performed, producing a sample with moderately cellularity. A large amount of fluid from the tracheal tube was noted. The dog was given a puff of albuterol prior to extubation. The dog was treated with oxygen, azithromycin, doxycycline, fluids, terbutaline, nebulization and coupage, and a dose of Lasix while admitted. The dog passed away on Wednesday morning. On necropsy, the dog is in good body condition (BCS 5/9), is mildly dehydrated (5-7%), and has mild tissue autolysis present. There is moderate dental calculus present over the premolar and molars of both arcades in the mouth, with moderate gingivitis. The lungs are dark red to dark pink in color, are mottled, and are congested with foam. The lungs are rubbery on palpation, and appear swollen. The pulmonary parenchyma of the left caudal lung lobe is diffusely dark red and congested with red tinged foam. There are no other lesions in any other organ system. On histopathology, the significant changes were observed in the lung tissues. An interstitial pneumonia, and alveolitis, subacute to chronic, necrotizing, severe diffuse, with fibrosis and alveolar hemorrhage was seen. Bacteriology results for lung tissue isolated two colonies of Alpha-haemolytic Streptococcus sp. . Virus isolation in the lung tissue was negative. The changes observed grossly on necropsy and on histopathology are consistent with **Acute respiratory distress syndrome (ARDS)** a condition of sudden respiratory failure due to the rapid accumulation of fluid and severe inflammation of the lungs. ARDS is thought to occur subsequently to trauma, or some other underlying medical condition, in which blood, fluid and tissue infiltrate the alveoli, and ultimately cause their collapse. In humans, there appears to be a genetic relation to development of this syndrome, but this has not been investigated in dogs. Oxygen toxicity is also seen in human medicine as a possible precursor to ARDS. There was no indication of an active infectious process in any of the tissues submitted for testing in this particular case. On follow-up, the owner of the dog in this case, who works in an animal shelter, mentioned that several of her personal animals had been diagnosed with Kennel Cough shortly after the initial dog died.

Dr. Brad Barlow

Canine Parvoviral Myocarditis

A litter of 5 Border Collie cross puppies, 4.5 weeks old and still nursing, along with the mother had been surrendered to a rescue/adoption organization. The original litter consisted of 7 puppies, 2 of which had died in the first 2 weeks. There was no known vaccination history. Over a 3 day period, 3 of the puppies died suddenly without any observed indication of illness. The last puppy to die was submitted for necropsy.

The Border Collie cross puppy examined was in good body condition and weighed 1.7 kilograms. White froth filled the trachea and all the lung lobes were edematous. The heart was rounded, measured 4 by 4 centimeters, both ventricles were dilated and the ventricular walls were thin. Milk filled the stomach.

COMPANION ANIMAL, CONTINUED

Histopathology findings revealed heart lesions characterized as severe, chronic, diffuse, fibrosing and lymphohistiocytic myocarditis and lung pathology consisting of diffuse, moderate, acute, alveolar edema and histiocytosis.

Canine Parvovirus was detected in the heart by immunohistochemistry testing.

The myocarditis form of parvovirus is generally seen in young puppies born to non immune bitches exposed to parvovirus in utero or as neonates. Puppies surviving initial infection may develop subsequent myocardial fibrosis. Death may occur due to cardiac arrhythmias or congestive heart failure. It is reasonable to presume that other puppies in this litter also died from parvoviral myocarditis. Due to improving vaccination status of dogs and the mature nature of parvovirus in the canine population, this form of the disease is now infrequently seen in our laboratory system.

Reference:

Greene, Craig E. Clinical Microbiology and Infectious Disease of the Dog and Cat. 1984. W. B. Saunders Company. pp 440 – 442.

Dr. Reggie A. Ridenhour

Cardiac Tamponade, Valvular Endocardiosis and focal myocarditis in a Weimeraner Dog

A 7.5-year-old neutered female Weimeraner with a 3 to 4 year history of a progressive systolic heart murmur (grade II/VI to IV/VI) acutely collapsed and died. An echocardiogram was never performed. Significant gross findings included: 600 ml hemopericardium, marked enlargement of the left auricle (twice the size of the right auricle) with a 2 cm tear, moderate mitral valve endocardiosis with corresponding jet lesions along the endocardial surface of the left atrium, hepatic congestion with a mild nutmeg pattern, multifocal fibrinous adhesions along the hepatic surface, 300 ml serosanguinous transparent fluid in each thoracic and abdominal cavity, and multifocal hemorrhagic pulmonary nodules that measure up to 1 cm. Histologically, there was mitral valve fibromyxomatous degeneration (**valvular endocardiosis**). **Suppurative myocarditis** with myocardial degeneration and necrosis, hemorrhage, fibrosis, a fibrin thrombus and intralesional bacterial colonies were identified at the rupture site. Also identified were pulmonary hemangiosarcoma and sinusoidal dilation and congestion (**chronic passive congestion**). Gram-positive bacteria were not identified in the heart. The cause of death was ultimately due to the cardiac tamponade. It was thought that the endocardiosis resulted in dilation of the left auricle which led to continued dilation and weakening of the cardiac muscle and adherence of bacteria to the damaged endothelium of the weakened wall followed by rupture.

Dr. Mahogany Caesar

COMPANION ANIMAL, CONTINUED

Feline

Toxoplasmosis in a Kitten

An 11-week-old female DSH kitten was found dead. Necropsy revealed thin body condition, moderate dehydration, slightly icteric mucous membranes and expansion of the mediastinum and costal pleura by moderate amounts of straw-colored gelatinous transparent material. Histopathologic review revealed lymphohistiocytic encephalitis and myocarditis and necrotizing interstitial pneumonia, splenitis, lymphadenitis and hepatitis. Associated with all lesions with exception of the heart were protozoal cysts and tachyzoites consistent with *Toxoplasma gondii*. **Toxoplasmosis** has been reported in nearly all warm-blooded animals. In felines, this infection is more commonly seen in strays, animal shelters, young kittens or immunocompromised cats, for instance those with feline immunodeficiency virus or feline leukemia virus. Many cats infected with *T. gondii* are asymptomatic. Clinical signs vary depending on the location of the infection. The large numbers of protozoal cysts (Figures 1 and 2) identified along with the multi-organ distribution make this a spectacular case of toxoplasmosis.

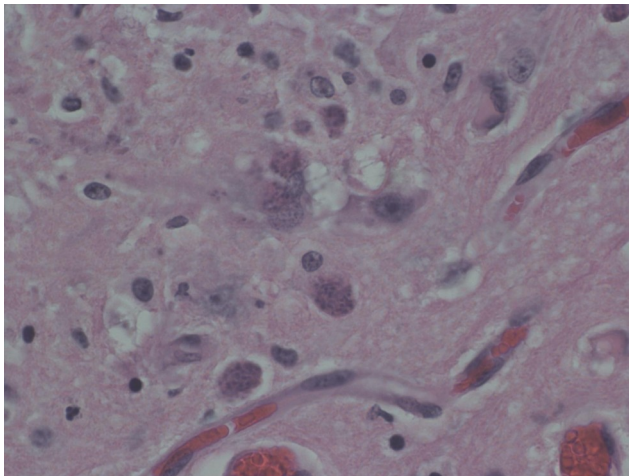


Fig 1. Brain. H&E. 60 x. Multiple tissue bradyzoites distributed throughout the parenchyma of the cerebral white matter.

COMPANION ANIMAL, CONTINUED

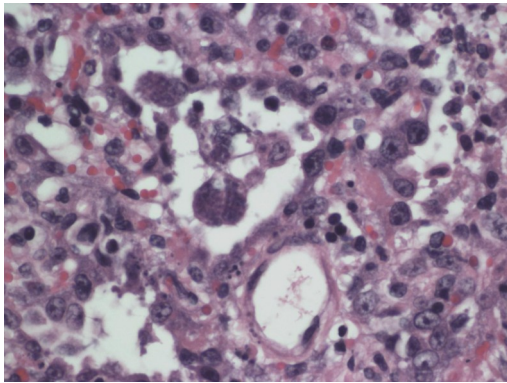


Fig 2. Lung. H&E. 60 x. Tissue bradyzoites in the pulmonary parenchyma resulted in interstitial destruction, necrosis and inflammation. Tachyzoites also present..

Dr. Mahogany Caesar

Exotics

Fenbendazole toxicity in a pigeon

An adult male white pigeon was presented to the laboratory for post mortem examination. The provided history stated the bird presented to the referring veterinarian a month prior in very thin body condition. The bird was treated with Fenbendazole for *Capillaria* sp. parasites. Recently, the bird began to have vomiting. On post mortem examination the bird weighed 198.9 grams. It didn't appear particularly thin in body condition. The crop was friable and the crop wall was red in color. Yellow plaques were present on the crop mucosa. Gram stain of an impression slide of crop content showed gram positive bacterial rods. No *Trichomonas* sp. organisms were seen. Histopathologic review revealed swelling of the lining epithelial cells of the crop, with some cells having enlarged nuclei, with occasionally seen syncytial epithelial cells. Additionally, there was diffuse necrosis of the intestinal crypts. The crypts are dilated, devoid of lining epithelium or lined by flattened epithelium, and contain necrotic cellular debris. The villi look short and blunt.

The pathologist felt the inflammation of the crop and intestine to be compatible with **Fenbendazole toxicity**.

J Am Assoc Lab Anim Sci. 2006 Nov;45(6):63-6. Mortality associated with fenbendazole administration in pigeons (*Columba livia*). Gozalo AS, Schwiebert RS, Lawson GW.

Dr. David Drum

COMPANION ANIMAL, CONTINUED

***Salmonella arizonae* in an adult Kingsnake**

The body of a 5 year old female albino Kingsnake was presented to the laboratory for post mortem examination. The morning of the snake's death, blood was noticed in the feces and the snake wouldn't eat.

On post mortem examination the snake weighed 361 grams and was in good body condition. Pink fluid was present in the stomach. Firmly clotted blood was tightly adhered to the intestinal mucosa. Blood was present from the beginning of the small intestine through to the cloacae. Multiple, unshelled, undeveloped eggs were present in the reproductive tract.

On histopathology, Intestine; basement membrane mineralization regionally extensive, chronic, severe, with multifocal. hemorrhage, Liver; sinusoidal heterophilia, diffuse, acute, mild to moderate, with intracytoplasmic coccobacilli. Stomach; luminal coccobacillary colonies. *Salmonella enterica* subsp. *arizonae* was isolated on bacterial culture of the intestine and bile.

Salmonella enterica subsp. *arizonae* is a common gut inhabitant of reptiles, with snakes serving as the most common reservoir. The organism has also been associated with lesions of osteomyelitis (bone infection) and diphtheritic necrotizing gastritis and tracheitis in snakes. There have been reported cases of illnesses in people who have kept reptiles as pets.

Histopathology showed the snake had diffuse metastatic calcification. This was likely secondary to kidney disease, which in turn could have been caused by the *Salmonella* infection.

Dr David Drum

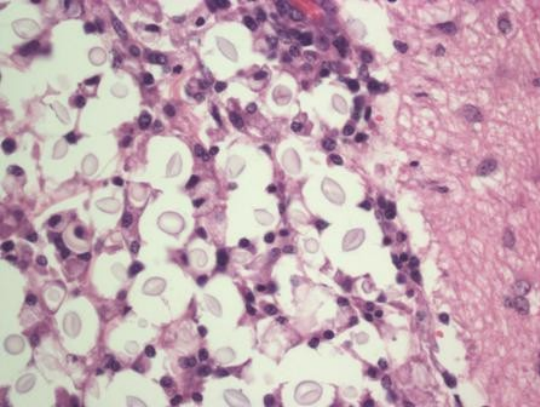
LIVESTOCK

Equine**Cryptococcosis in an Icelandic Mare**

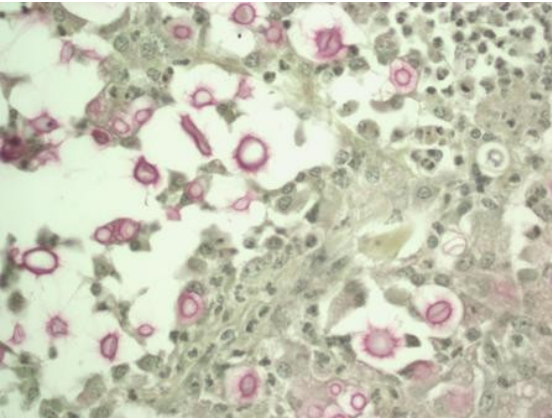
A 14 year old Icelandic mare was euthanized due to progressive weight loss for the past 45 days and ataxia of all limbs that improved after treatment with IV fluids and DMSO with a relapse of more pronounced clinical signs 10 days after each of the two treatments. She did not respond to the third treatment and her condition continued to deteriorate. Vaccinations were current. She was euthanized and submitted for necropsy.

Necropsy examination of the brain and spinal cord was largely unremarkable. Histopathology examination of the brain and spinal cord identified marked pyogranulomatous meningoencephalitis and mild meningomyelitis with intralesional yeasts consistent with *Cryptococcus neoformans*.

LIVESTOCK, CONTINUED



Photomicrograph of cerebrum and meninges with inflammation and yeasts consistent with *Cryptococcus neoformans*. H & E, 400 X.



Photomicrograph of cerebrum and meninges with inflammation and yeasts consistent with *Cryptococcus neoformans*. Mucicarmine, 400 X.

Cryptococcal meningitis is a rare condition in horses. It occurs more frequently in small domestic animals, especially cats. Cryptococcal infections in the horse typically involve the respiratory tract alone, specifically the nasal sinuses. While it is unknown how the yeasts enter the central nervous system, it is thought they pass from the nasal sinuses via the cribriform plate into the CNS and then circulate in the cerebrospinal fluid. Another proposed route is hematogenous spread via the lungs. *Cryptococcus neoformans* is normally found worldwide in the feces of birds, in the soil, and on certain plant species. In humans, *Cryptococcus neoformans* is usually acquired by inhalation. Infected individuals are often immunosuppressed. The respiratory tract is considered the primary route of infection. The most definitive diagnostic test for Cryptococcal meningitis is via cerebrospinal fluid analysis with visual identification of the organisms.

Dr Kim Hagans

LIVESTOCK, CONTINUED

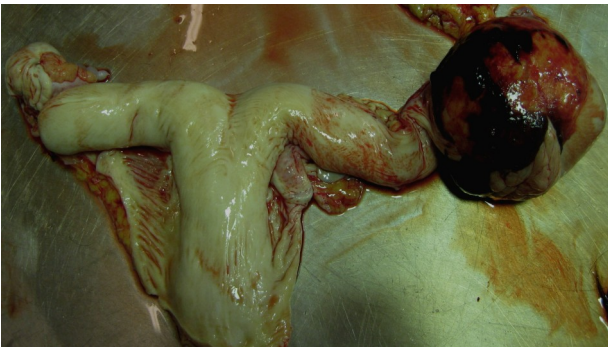
Equine Ovarian Hematoma

A 3 year old Arabian mare was reported to be normal one day and very lethargic the next day. The veterinarian examined her and on physical exam she had tachycardia (88 bpm heart rate), was wobbly and had a PCV of 18. The veterinarian palpated the mare and noted her left ovary was enlarged. The mare died early the next morning.

The 340kg 3 year old Arab mare was submitted for post mortem examination. External exam revealed white mucous membranes and a Body Condition Score of 5/9. **Hemoabdomen** was noted and all tissues were pale. The left ovary was approximately 10 cm in diameter with hematoma formation. An area of ovarian epithelium was eroded and had small areas of clot formation. The hemorrhage appeared to originate from the ovulation fossa. Histopathology findings included: Left ovary: Marked subacute and acute hemorrhage (hematoma formation) with surface hemorrhage and fibrin deposition. Bone marrow: Moderate erythroid and megakaryocyte hyperplasia.

The **hematoma** associated with the left ovary had lamellar fibrin deposition consistent with recurrent hemorrhage. There was luteinization of the wall associated with the hemorrhage and there were medium caliber vessels and small vessels at the margin on the hemorrhage that had been disrupted.

This is a very unusual case as most often ovarian hematomas regress over time and are of little consequence. There was lamellar fibrin consistent with recurrent hemorrhage. The bone marrow was mildly hyperplastic consistent with a regenerative anemia.



Dr Kim Hagans

LIVESTOCK, CONTINUED

Equine Neonatal Isoerythrolysis

A 6 day old mixed breed foal died after a 24 hour period of icterus and hypothermia. The dam was primiparous and was accidentally bred in pasture by a yearling. The mare had a retained placenta. The foal was seen nursing and acting normal the first 4 days of life. At necropsy the tissues were severely icteric; no other significant gross lesions were present. Histopathology revealed peracute pulmonary arteriolar fibrin thrombi, marked acute centrilobular hepatocellular degeneration and necrosis (hypoxic damage) with marked intracanalicular bile plugs, marked acute splenic congestion, moderate peracute tubular epithelial degeneration and necrosis and wide spread erythrophagocytosis. Differentials for icterus in a neonatal foal are neonatal isoerythrolysis (NI), sepsis, meconium impaction, and liver failure. The histological lesion of wide spread erythrophagocytosis and associated hypoxic changes are most consistent with **neonatal isoerythrolysis**. This syndrome is usually seen in multiparous mares due to a previous exposure to the sire's red blood cells and the development of anti-erythrocyte antibodies, which are secreted in the mare's milk. It is interesting that the mare was primiparous with no history of blood transfusion; perhaps there was a placental abnormality in a previous accidental breeding to the same yearling that allowed leakage of fetal red blood cells into maternal blood.

Dr Jennifer Haugland

CE ATTENDANCE

Dr. Brad Barlow attended the USDA Foreign Animal Disease Diagnostic Course held at the Plum Island Animal Disease Center, Plum Island, NY on November 14-18, 2011

Dr. Kim Hagans attended the Georgia Veterinary Medical Association conference May 30-June 2, 2012.

Beverly Wood, Molecular Diagnostics Section Supervisor, attended the Biocontainment Basics and Zoonotic Disease Training Course held May 1-2, 2012 at the National Centers for Animal Health, in Ames, IA. The course was developed by the Center for Excellence for Emerging and Zoonotic Animal Diseases (CEEZAD) with funding from the Department of Homeland Security (DHS) and focused on biocontainment BSL-3 practices, laboratory safety, handling packages containing unknown agents, information about selected foreign animal and zoonotic diseases that are listed as terrestrial threats to the United States and its territories, and detecting these agents.

Katheryn Schmidt, Laboratory Safety Officer, attended the NC Industrial Commission Safety Leadership Workshop in Fayetteville, NC from March 6-8, 2012.

DEPARTMENTAL NEWS**CE ATTENDANCE**—CONTINUED

Dr. Tahseen Aziz, Avian Pathologist, attended the 2012 Western Poultry Disease Conference in Scottsdale, Arizona from April 1-5, 2012.

Cynthia Nipper and Faye Coombs, Histopathology technicians, attended the NC Society of Histopathology Technicians Meeting & Workshop in Raleigh, NC from April 26-28, 2012.

ROLLINS LABORATORY**New Hires**

Laura Marley-Trotter; started 2/01/2012 as a Medical Laboratory Technician II

Dr. Chad Cecil; started 3/07/2012 as a Laboratory Medical Specialist

Jeri Smart; started 5/07/2012 as a Processing Assistant III

Resignations

Gina Lombardi, Medical Laboratory Technologist II, Resigned 7/06/2012 with over 10 yrs of service, the majority of which was spent in the Molecular Diagnostics Section.

J. Renee Atkinson, Laboratory Assistant, resigned her position effective 7/13/2012.

Milestones

The following Rollins employees have received service awards to date in 2012:

Dr. Mahogany Wade Caesar – 5 years

Aisha Phipps – 10 years

Sandy Murphy – 20 years

Kim Howle – 20 years

Dr. Karen Post – 20 years

Jennifer Pruitt – 35 years

ELKIN LABORATORY

Dr Brad Barlow was a presenter at the NC Department of Agriculture, HAZWOPER Training on March 22, 2012 in Raleigh, NC. Dr. Barlow gave a presentation on the subject of Foreign Animal Diseases.

Directory

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[Dr. Tahseen Abdul-Aziz](#)

[Dr. Peter Moisan](#)

[Dr. Steven Rushton](#)

[Dr. Alison Tucker](#)

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[Dr. Jennifer Haugland](#)

[Dr. Stacy Robinson](#)

[Dr. Mahogany Wade](#)

Veterinary Microbiologists

[Dr. Karen Post](#)

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[Dr. Chad Cecil—Virology](#)

[Sandy Murphy—Bacteriology](#)

[Mary Horne—Histopathology](#)

[Jennifer Pruitt—Serology](#)

[Beverly Wood—Molecular Diagnostics](#)

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Dr. Jennifer Haugland	NCDA&CS Veterinary Diagnostic Laboratory System